Environment Canada Environnement Canada

Guidelines for Surface Water Quality

Vol. 2 Organic Chemical Substances

HEXACHLOROBUTADIENE

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Abstract

A literature survey was carried out on the toxic effects of hexachlorobutadiene on human health, aquatic life, plants and livestock. The information is summarized in this publication. From it, maximum hexachlorobutadiene concentrations in water at which toxic effects will not appear are recommended.

Résumé

On a effectué une étude de la documentation portant sur les effets toxiques de l'hexachlorobutadiène sur les humains, les organismes aquatiques, les plantes et le bétail. Ces données sont résumées dans cette publication. En se basant sur ces données, on propose des valeurs des concentrations maximales d'hexachlorobutadiène dans l'eau auxquelles on n'observe pas d'effets toxiques.

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RECOMMENDED WATER QUALITY OBJECTIVES FOR HEXACHLOROBUTADIENE

Uses	Recommended objective
Raw public water supply	No drinking water guideline, therefore no recommended limit
Aquatic life and wildlife	0.1 μg/L
Agricultural water supply	No recommended limits, insufficient information
Recreation and aesthetics	No recommended limits
Industrial water supplies	No recommended limits

Guidelines for Surface Water Quality

Vol. 2. Organic Chemical Substances. Hexachlorobutadiene

SOURCES, OCCURRENCE AND CHARACTERISTICS

Production and Uses of Hexachlorobutadiene

Hexachloro-1,3-butadiene, commonly referred to as hexachlorobutadiene, is abbreviated to HCBD. It does not occur naturally, and therefore any input into the environment must be man-made.

The indirect production of HCBD is considerable, since it is found in wastes as a by-product of chlorinated hydrocarbon manufacturing. About 99% of total HCBD formation in the United States results from the production of carbon tetrachloride, trichloroethylene and tetrachloroethylene. In 1972 in the United States, the secondary production of HCBD associated with these chemicals was of the order of 3.31×10^6 to 6.58×10^6 kg (Mumma and Lawless 1975).

Estimates of the percentage of HCBD generated during the production of carbon tetrachloride, trichloroethylene and tetrachloroethylene are listed in Table 1. If these estimates were to hold for the 1973 world production of these chemicals (McConnell *et al.* 1975), then approximately 10×10^6 to 20×10^6 kg of HCBD would have been generated in the world in 1973.

Table 1. Estimated HCBD Production in Chemical Manufacturing

Chemical	HCBD by production (percentage)
Tetrachloroethylene	0.5 to 1.0
Trichloroethylene	0.3 to 0.63
Carbon tetrachloride	0.1 to 0.25
Chlorine	<0.0003

Source: Mumma and Lawless 1975. Based on 1972 production in the United States and includes HCBD wastes, effluents, emissions and products.

Similarly for Canada, the estimated 1979 output of HCBD as a by-product of the manufacturing of these three chemicals was about 160 000 to 340 000 kg. Based on total production capacity, the potential Canadian production rate is of the order of 200 000 to 400 000 kg per year (Acres Consulting Ltd. 1981). In Canada in 1979, the production of these three compounds was limited to sites by the lower Great Lakes and the St. Lawrence River: Sarnia, Georgetown, Cornwall and Shawinigan (Environment Canada unpublished 1979).

The recovery and purification of HCBD generated as a process by-product apparently ceased in the United States in 1970, although at least one company is still listed as a producer (TSCA/ITC 1978; Environment Canada unpublished 1979). Subsequently, importation from Europe has been the source of this chemical. In 1974, the estimated importation into the United States was between 90 000 and 227 000 kg (Mumma and Lawless 1975). Information on importation into Canada is not available, and although Canadian distributors do exist, importation in industrial quantities is uncommon (Environment Canada unpublished 1979). Possible importation by a Canadian subsidiary of a foreign producing or distributing company for internal use was not included in this evaluation.

The direct production of HCBD is known to take place by several methods, such as chlorination and dehydrochlorination of hexachlorobutene, chlorination of polychlorobutanes, and catalytic chlorination of butadiene. It is not known to have been directly produced in commercial quantities in the United States or Canada (IARC 1979; Environment Canada unpublished 1979). In general, worldwide information concerning manufacturing sites and production rates is not available.

Although 19 potential or actual uses of HCBD have been identified worldwide (Table 2), Canadian usage is unknown (Environment Canada unpublished 1979). In the United States the most important use identified is the recovery of chlorine from waste gases. It is also utilized as an intermediate in the production of rubber compounds and lubricants, and as a fluid for gyroscopes, as a component of hydraulic fluids, as a heat transfer liquid in electrical transformers and as a solvent for elastomers (Mumma and Lawless 1975; Hawley 1981). The quantities used in these processes are not known.

Table 2. Actual and Potential Uses of Hexachlorobutadiene

Solvent for elastomers
Component of hydraulic fluids
Heat transfer fluid
Component of transformer fluid
Wash liquor for removing hydrocarbons (C_4 and higher)
Recovery of chlorine from effluent gases at chlorine-producing facilities
Fluid in gyroscopes
Intermediate in the production of certain specialized lubricants
Solvent for chlorination of waste gases containing low boiling point unsaturated hydrocarbons
Growth stimulant for plants
Cutting lubricant for metal machining
Solvent used in the production of copper phthalocyanine pigment
Polymerizer of fire-resistant polyamides
Agent for rapid setting and rapid demolding of nonelastic polyurethanes
Removing $FeCl_3$ from chlorine gas
Fungicide and pesticide in vineyards
Component of a pottery-like coating that sets at room temperature
Chemical intermediate in the production of certain rubber compounds
Solvent for chlorination reaction

Source: Environment Canada unpublished 1979

Hexachlorobutadiene has been employed elsewhere in the world—USSR, France, Italy, Greece, Spain and Argentina—as a fungicide and/or pesticide in vineyards. The USSR expended 600 000 to 800 000 kg for this purpose (Mumma and Lawless 1975), but it does not appear to have been used as an agricultural chemical in North America, and certainly not in Canada (Environment Canada unpublished 1979).

Chemical Characteristics

The properties of HCBD are summarized in Table 3.

Table 3. Properties of Hexachlorobutadiene

Physical description	Clear, colourless, nonflammable liquid
Odour	Faint turpentine-like odour
Molecular formula	$CCl_2:CClCCl:CCl_2$
Molecular weight	260.76
Melting point	-21°C
Boiling point	215°C
Specific gravity	1.68[1]
Vapour pressure	0.15 mm Hg at 20°C 22 mm Hg at 100°C[1]
Solubility in water	5 mg/L at 20°C[2] 2.4 mg/L at 20°C[3]
Log octanol/water partition coefficient	3.74 (calculated)[4] 1.82 (measured)[5]
Water/air partition coefficient	0.97 at 20°C

[1] Hawley 1981
[2] TSCA/ITC 1978
[3] Leeuwangh *et al.* 1975
[4] USEPA 1980
[5] Dec *et al.*, MS cited in USEPA 1980

HCBD in the Environment

One of the most important factors in the release of HCBD into the environment is the handling and disposal of wastes containing HCBD formed as a manufacturing by-product. Often waste holding areas—landfill and open-pit or lagoon storage—are the most significant emission sources. Waste loading and transfer areas, when employed, are also important. Incineration of wastes generally reduces net HCBD losses. Process wastewater can also be an important loss pathway (Li *et al.* 1976).

The handling of wastes containing HCBD from the chlorinated hydrocarbon manufacturing industry is not well documented for Canadian producers. One Canadian company indicated that some of this material is recycled or incinerated and that some is disposed of in an impervious underground cavern or at a landfill site (Bonner and Meresz 1981). In the United States in 1971, the distribution of HCBD in such industrial wastes was 98% in solid waste (a tarry substance high in hexachlorobenzene as well), 1% as airborne emissions, and 1% as aqueous effluent. This may not be representative of more recent technology and plant operation, and it is not known whether it is applicable to Canadian manufacturers.

Due to the tarry nature of the waste material, direct dispersal of solids (e.g. windblown dust) is likely to be negligible. Direct and indirect (volatilization and solubilization from solid wastes) airborne and waterborne dispersion are therefore the primary pathways for the entrance to and spread throughout the environment. The details of movements through the ecosystem and the ultimate fate of dispersed HCBD are unknown.

There is little information on the levels of HCBD present as an impurity in the primary manufactured products of carbon tetrachloride, trichloroethylene and tetrachloroethylene. One Canadian chemical producer has indicated that HCBD levels in their solvents are well below 1 ppm (Bonner and Meresz 1981). If this level of contamination is indicative of all production, then it represents at worst (assuming 1 mg/kg) about 60, 1100

and 3000 kg of HCBD distributed with the entire annual Canadian, American and world production, respectively, of chlorinated hydrocarbon solvents. Losses of HCBD resulting from the use and disposal of these compounds are unknown.

It has been suggested that HCBD may be a commonly occurring contaminant in the watercourses of the Netherlands (Goldbach *et al*. 1976). It has been detected in some widely scattered areas of North America as well. There are some data for fish in the Great Lakes, but none for sediment or water concentrations, the latter being below detection limits. For Lake Superior, Swain (1978) and Swain *et al*. (1978) reported very limited and scattered occurrence of HCBD in 5 out of 54 species examined with mean levels ranging from about 0.5 to 1.0 mg/kg, essentially at the detection limit. Kuehl *et al*. (1981) in a much less extensive study did not detect HCBD in fish sampled in Lake Superior, Lake Michigan and Lake St. Clair but did find levels in Lake Ontario fish ranging from 1.7 to 2.5 μg/kg.

Clearly, further work is required to clarify the distribution of HCBD in the environment of North America. The general case for organochlorine compounds, however, provides some illumination. The atmospheric deposition of PCB's (polychlorinated biphenyls), which are organochlorine compounds that have some similar characteristics but with much lower vapour pressures, represents the principal input into the upper Great Lakes and a significant source to the lower lakes. In remote locations, atmospheric input is the only method of transport of these materials into the area. For some trace organics the vapour phase is the major proportion of the airborne load, although adsorption to aerosols can be significant (Eisenreich *et al*. 1981). Therefore, despite its low vapour pressure, volatilization and aerial transport may be an important pathway for dispersal of HCBD in the environment (McConnell *et al*. 1975). Other than the observation that HCBD levels in air, when detectable, range from 0.003 to 460 $\mu g/m^3$ (typically less than 5 $\mu g/m^3$) in the vicinity of chemical plants where HCBD is a known by-product (Li *et al*. 1976), little information on airborne HCBD is available.

Levels and Interactions in Water and Sediment

Data for Canada are very limited. Bonner and Meresz (1981) reported that HCBD was found at 5 out of 31 sites and in 5 out of 88 samples during a survey of the aqueous effluent from petrochemical plants along the St. Clair River. In four of these effluents the level was not quantified but was estimated at between 10 and 100 μg/L, while in the fifth it was estimated at between 0.05 and 0.1 μg/L. Hexachlorobutadiene was detected in river sediments at one of the three sites examined; 50 mg/kg (wet) was the value determined in an approximate quantification. It was not detected in St. Clair River water samples (detection limit of 0.05 to 0.1 μg/L).

In Europe, HCBD has been found at levels as high as 0.27 μg/L (IARC 1979). Levels in drinking water in the United States are, when detectable, usually less than 0.2 μg/L (Benedek and Bancsi 1977). In southern Louisiana, levels in water and sediment on a regional overview basis ranged from 0.9 to 1.9 μg/L and from 2.16 to 790 μg/kg (wet), respectively, when present above the detection limit. In this same region in the immediate vicinity of an industrial source of HCBD, water levels ranged from 0.04 to 4.7 μg/L (typically less than 1 μg/L), and sediment levels ranged from 190 to 1080 μg/kg (wet) (Laseter *et al*. 1976). Process wastewater from industries where HCBD is known to be a by-product contained from 0.04 to 224 μg/L HCBD when detectable (Li *et al*. 1976). Levels in water in the Rhine River in the Netherlands ranged from 0.05 to 0.20 μg/L (Goldbach *et al*. 1976).

Sediment levels of HCBD are typically two or three orders of magnitude higher than the corresponding water concentration, both in natural and in laboratory situations (Laseter *et al*. 1976; Goldbach *et al*. 1976; McConnell *et al*. 1975). Leeuwangh *et al*. (1975) generated an equation which provided a rough description of the water-sediment partitioning of HCBD observed in their laboratory experiment:

$$a = 200c^{0.9}$$

where a = weight of HCBD sorbed per unit weight of soil
c = HCBD concentration in solution at equilibrium.

This water-sediment distribution is to be expected, as due to its hydrophobic nature, HCBD binds tightly to the organic (humic) component in the system (Khan 1980). Concentrations of HCBD in water would probably be stabilized by the reservoir of material in the bottom sediments. This has been observed under experimental conditions (Laseter *et al*. 1976). The stabilization capacity of any system is largely dependent on organic content (Leeuwangh *et al*. 1975). Karickhoff *et al*. (1979) noted that sorption of hydrophobic pollutants onto sediments is a function of the octanol/water partition coefficient of the compound in question. Other organic components, including biota, in the aquatic ecosystem may participate in this stabilizing action as well. This depends on the rate of interchange; slow rates of interchange contribute little to the stabilizing action.

Persistence and Abiotic Degradation

Hexachlorobutadiene is generally considered to be a stable and persistent environmental pollutant that is highly resistant to chemical, biological and physical degradation (Fishbein 1979). It is not reactive with acids and alkalis and is extremely resistant to hydrolysis. However, it does react with ozone and the OH-radical, exhibiting a half-life of less than one day (TSCA/ITC 1978). Photochemical degradation may be an important pathway for reducing HCBD levels in the environment; for example, the half-life of HCBD vapour exposed to sunlight is about one week (Korte 1979; TSCA/ITC 1978). On the other hand, the breakdown of HCBD in water appears to be slow, and is probably due to the formation of complexes with organic constituents and sorption onto particulates. Although there is evidence that complexation with humic substances in wet soil and at the surface of water bodies may promote ultraviolet catalyzed hydrolysis, the distribution of HCBD in the water column and in the sediments, coupled with the limited penetration capability of ultraviolet light, probably limits the influence and effectiveness of this reaction (Khan 1980).

The quantification of the persistence and abiotic degradation of HCBD in the aquatic environment is not currently possible due to a dearth of information.

RATIONALE

Raw Public Water Supplies

As no guideline for drinking water has been proposed by Health and Welfare Canada, a guideline for raw public water supplies has not been formulated.

Aquatic Life and Wildlife

Levels in Aquatic Biota

Plants

Very little information is available on HCBD levels in aquatic plants. In an area known to be contaminated with HCBD, where water levels ranged from 0.1 to 1.2 µg/L (mean of 0.6 µg/L) over ten months, single samples of three plant species—(submerged) *Chara* sp. and *Najas* sp. and (emergent) *Eleocharis* sp.—contained 12, 9 and 20 µg/kg (wet weight), respectively. This represents 20, 15 and 33 times, respectively, the mean water concentration during the sampling program. During this time, the mud substrate had a mean HCBD concentration of 440 µg/kg, representing a concentration factor of 730. In a seven-day laboratory experiment, the green alga *Oedogonium cardiacum* accumulated a maximum of 2700 µg/kg of HCBD when exposed to 16.9 µg/L in water, a bioconcentration factor of 160 (Laseter *et al*. 1976).

Invertebrates

Amphipod and oligochaete samples taken from Lake Ontario near the mouth of the Niagara River had concentrations of HCBD of 4 to 13 µg/kg (mean 8 µg/kg) and 1 to 4 µg/kg (mean 2 µg/kg), respectively. Niagara River water (30 weekly samples) had a range of 0.2 to 1.9 ng/L (ppt) (mean 0.5 ng/L). Lake Ontario sediments (45 samples lake-wide) had a range of 0.2 to 100 µg/kg (mean 0.25 µg/kg) (Oliver personal communication).

Water levels were 0.6 µg/L and mean sediment levels were in the vicinity of 550 µg/kg (wet weight), at a site of known HCBD contamination in Louisiana. An unidentified dragonfly nymph was reported to have had a level of 375 µg/kg. Snails, *Physa* sp., had a mean level of 345 µg/kg, whereas crayfish, *Procambarus* sp., had a mean level of 997 µg/kg. This represents bioconcentration factors of 565, 575 and 1780, respectively. Uncontaminated crayfish were placed in cages and held in a pond where the mean water HCBD level was 4.6 µg/L. The long-term (51 days) mean accumulation was 405 µg/kg, representing a bioconcentration factor of about 88. In the depuration portion of this experiment, crayfish were exposed to HCBD for 10 days, then placed in clean water; over 90% of the accumulated HCBD was eliminated after 12 days (Laseter *et al*. 1976).

Water concentrations in the region of 0.13 µg/L HCBD have been observed in the Ketelmeer, a lower portion of the Rhine River in the Netherlands. From this area molluscs, *Lymnaea peregra* and *Sphaerium* sp., exhibited mean HCBD levels (with shells excluded) of 0.85 and 2.41 mg/kg. This represents concentration factors of 6500 and 18 500, respectively. Oligochaetes, mainly *Limnodrilus* sp., had a mean level of 0.28 mg/kg, indicating a bioconcentration factor of 2100. Organic detritus collected at the same time had a level of 0.21 mg/kg, representing a concentration factor of 1600 (Goldbach *et al*. 1976).

Fish

Sunfish, *Lepomis* sp., from the lower Mississippi River in the United States, which is an area known to be contaminated, exhibited a mean HCBD concentration (whole body, wet weight) of 600 µg/kg. This is a concentration of about 1000 times the mean water level of

0.6 μg/L and is greater than the sediment concentration factor of about 440. The mosquito fish, *Gambusia affinis*, had mean HCBD levels of 890, 817 and 16 205 μg/kg in water that averaged 0.6, 0.56 and 4.6 μg/L, respectively. This represents concentration factors of 1480, 1460 and 3525, respectively, in an area where the sediment exhibited concentration factors of 440, 1200 and 110, respectively (Laseter *et al*. 1976). For the same general area, fish HCBD levels in a variety of species have been reported to range from less than 0.005 mg/kg to 4.59 mg/kg (mean of 0.81 mg/kg) (Yurawecz *et al*. 1976) and from 0.01 to 1.2 mg/kg (Yip 1976). Water or sediment levels were not reported in these latter two papers.

Swain (1978) and Swain *et al*. (1978) noted that out of 54 species of fish collected in Lake Superior, five species—lake trout, lake herring, American smelt, longnose suckers and walleye—had HCBD present, all at a level very near the quantification limit of about 1 mg/kg wet weight. The occurrence of HCBD was sporadic, with less than 1% of the more than 10 000 fish that were examined having HCBD present. When detected, HCBD levels in a lipid extract were about one order of magnitude higher than the whole body burden. Siscowet lake trout, a subspecies known to have exceptionally high body lipid levels, and in this study sampled only below a depth of 180 m, did not have detectable body burdens of HCBD. The Lake Superior area is removed from direct industrial influence. There is no evidence of direct aqueous HCBD input and HCBD levels in water are below a quantification limit of 0.1 μg/L; in fact, the total organochlorine residue level, of which HCBD is but a fraction, is about 0.005 μg/L in these waters.

On the other hand, Kuehl *et al*. (1981) reported that HCBD was not found in Lake Superior and Lake Michigan lake trout, but that it was detectable in fish in Lakes Erie and Ontario. Suns (personal communication) reported that HCBD was not detected (detection limit 2 μg/kg wet weight) in young of the year spottail shiners collected at seven sites between the Detroit River and the upper St. Lawrence River. In the latter two cases, relatively small sample sizes may be a contributing factor to the lack of detection. The lipid levels in the fish and their age may also be involved.

Oliver (personal communication) analyzed ten rainbow trout from the Gananaska River spring run (emptying into Lake Ontario) and reported a range of 0.06 to 0.3 μg/kg (mean 0.2 μg/kg).

Fields Brook, a tributary stream to Lake Erie located in Ashtabula, Ohio, had a water HCBD level of 22 μg/L (Konasewich *et al*. 1978). Fish samples from several species in another sampling year had HCBD levels ranging from 811 to 3250 μg/kg (Kuehl *et al*. 1981).

In the Ketelmeer, an inland water area of the Netherlands fed by the Rhine River, water HCBD levels were found to be about 0.13 μg/L. The mean level of 29 resident fish of different species (pike perch, perch, pike, tench, common bream, white bream and roach) was 0.75 mg/kg (wet weight, whole fish) with a range of 0.11 to 2.04 mg/kg. This represents a bioconcentration of over 5700 times the water level. No organ-specific accumulation was detected (liver, ovary and kidney), nor was any relationship found between body lipid content and HCBD content. In a connected area, the Ijsselmeer, the mean HCBD level of 20 fish (pike perch, eel, smelt, common bream and roach) was 0.038 mg/kg with a range of 0.008 to 0.136 mg/kg. This represents a bioconcentration factor of about 630 times the water level (≃0.06 μg/L) (Goldbach *et al*. 1976).

Accumulation and Elimination of HCBD in Aquatic Biota

From the previous observations it can be safely concluded that HCBD is found in aquatic organisms at levels higher than those found in the water in which they live. The mechanisms producing this phenomenon, however, are not clear. Adsorption and complexation with organics is an important factor in explaining higher HCBD levels in the non-living organic component of the aquatic system compared with water. It may be that the elevated levels found in organisms are primarily a function of physical-chemical processes operating in conjunction with the number of binding sites and the lipid levels. Goldbach *et al*. (1976) reported a significant correlation between HCBD content and fat content for several fish species.

Depuration of HCBD by goldfish and crayfish has been observed, with a half-life in HCBD-free water of about six and four days, respectively (Goldbach *et al*. 1976; Laseter *et al*. 1976). It is not known whether this elimination is a result of mass balance equilibrium, selective excretion, or biodegradation. Hexachlorobutadiene is considered to be highly resistant to biological breakdown (Korte 1979, Fishbein 1979), although HCBD, at concentrations of 5 and 10 mg/L, was completely degraded by a microbial culture in less than one week in a standard biodegradability test (Tabak *et al*. 1981). The applicability of these results to natural conditions is very questionable (Liu 1982 personal communication).

Biomagnification, or the increasing concentration of HCBD through the food web, has not been observed to any significant extent either in the field (McConnell *et al*. 1975; Goldbach *et al*. 1976) or in the laboratory (Laseter *et al*. 1976; Macek *et al*. 1979). This may be due to the relatively rapid depuration rates noted earlier.

Toxicity to Aquatic Organisms

Plants

No data on the toxicity of HCBD to phytoplankton or macrophytes have been found in the literature.

Invertebrates

Nosov and Kolosova (1979) reported that 100% mortality occurred within 4 h when *Daphnia magna* was exposed to 5000 µg/L HCBD; 50% mortality occurred in 2.4 days at a concentration of 2000 µg/L. There was 50% mortality for the crayfish *Procambarus clarki* in 57 h and 100% within 12 days at a concentration of 2700 µg/L. When HCBD in oil was injected into crayfish, they exhibited 30% mortality within 6 days at a dose of 34 µg/g of body weight (Laseter *et al.* 1976). The isopod *Asellus aquaticus* and the snail *Lymnaea stagnalis* demonstrated 96-h LC_{50} values (concentrations producing 50% mortality in 96 h) of 130 and 210 µg/L, respectively, in static tests (Leeuwangh *et al.* 1975).

Fish

Acute toxicity testing with HCBD has generated a number of mortality estimates for various fish species: the 96-h LC_{50} (static) for goldfish, *Carassius auratus*, is 90 µg/L (Leeuwangh *et al.* 1975); the 96-h LC_{50} (flow-through) for fathead minnows, *Pimephales promelas*, is 102 µg/L (Holcombe and Phipps 1980); the 48-h LC_{50} (flowthrough) for zebra fish, *Brachydanio rerio*, is 1000 µg/L (Slooff 1978); and the 96-h LC_{50} (flowthrough) estimates for bluegills, *Lepomis macrochirus*, and rainbow trout, *Salmo gairdneri*, are 326 and 320 µg/L, respectively, (USEPA unpublished cited in USEPA 1980). Bass, *Micropterus salmoides*, and mollies, *Poecilia latipinna*, were tested in static brackish water (1.0 and 2.5 ppt salinity, respectively). The bass had 24- and 48-h LC_{50} values of 2500 and 4300 µg/L, respectively, and the mollies had 26- and 30.5-h LC_{50} values of 4200 and 4500 µg/L. The projected 96-h LC_{50} is about 1600 µg/L. Some of the symptoms of exposure of fish to acutely toxic levels of HCBD include partial immobilization, loss of balance, and erratic swimming and gasping. These are suggestive of nervous system damage (Laseter *et al.* 1976; Laska *et al.* 1978a and b).

There are few data on fish concerning chronic toxicity testing with HCBD. An embryo-larval test with fathead minnows indicated a threshold chronic toxicity of 9.3 µg/L with confidence limits of 6.5 to 13.3 µg/L (USEPA unpublished data cited in USEPA 1980). Laska *et al.* (1978b) reported elevated corticosteroid levels, a generally accepted indication of stress, in bass exposed to 3.4 (the lowest concentration tested) and 32 µg/L of HCBD for ten days. A test based on an alteration in respiration frequency indicated that rainbow trout were able to sense HCBD in water at a level of 50 µg/L (initial concentration) within 24 h (Slooff 1978).

HCBD in Wildlife

There is little information on which to base an assessment of the effects of HCBD on wildlife. Schwetz *et al.* (1974) studied the effects of HCBD on the Japanese quail *Coturnix coturnix japonica*. A diet containing 0.3 to 30 mg/kg of HCBD had no effect on body weight, demeanor, food consumption, egg production, percent fertility and hatchability of eggs, eggshell thickness, or survival of the hatched after 90 days of feeding. No signs of toxicity were noted, nor could any changes in organs or tissues be found in gross and histological examinations. Based on average food consumption, the highest application rate was of the order of 5 to 6 mg/kg per day.

Recommended Limit for Aquatic Organisms and Wildlife

It is apparent that HCBD is highly toxic to aquatic organisms, with acute effects on fish and invertebrates occurring at water concentrations ranging from 90 to 1000 µg/L. Chronic effects on fish are detectable at a mean level of 9.3 µg/L. Knowledge of the consequences of HCBD in the aquatic environment is not sufficient to enable a reasonable estimate of a level at which all aquatic organisms would be safe. However, by applying an arbitrary safety factor of 0.01, a provisional value for a no effect level can be generated (Roeder 1979). Therefore the levels of HCBD should not exceed the 0.1 µg/L to protect aquatic life and wildlife.

Livestock Watering

HCBD in the Mammalian Body

No information has been found on the presence in or toxicity to large domestic animals. However, some information on animal-derived food products is available. In Germany, HCBD has been detected in condensed milk (4 µg/kg), chicken feed (39 µg/kg), egg yolk (42 µg/kg) and chicken by-product meal (2 µg/kg). It was not stated whether the chicken feed (grain) tested was that fed to the chickens from which the eggs and meal were produced (Kotzias *et al.* 1975). In Great Britain, fresh milk and locally produced butter have been reported to contain 0.08 and 2.0 µg/kg of HCBD (McConnell *et al.* 1975). In the lower Mississippi basin of North America, one milk sample collected within 40 km of a plant producing

HCBD contained 1.32 mg/kg, but other milk samples as well as egg samples did not have detectable levels (Yip 1976; Yurawecz *et al*. 1976).

Persistence and excretion or degradation are essentially unknown. In laboratory tests with rats, little is accumulated in most tissues. Hexachlorobutadiene may be conjugated with compounds containing mercapto(SH), especially glutathione, and excreted in bile and urine (Harleman and Seinen 1979; Davis *et al*. 1980).

Although in these reports HCBD has not been found regularly as a contaminant in animal produce (e.g. milk), its widespread sporadic occurrence indicates that it must be considered a general contaminant with substantial mobility in the ecosystem.

The following section discusses the toxic effects of HCBD on small laboratory mammals, indicating which organs may be affected and the type of response to HCBD that can be expected in livestock watered with HCBD-contaminated water.

Toxic Effects of HCBD to Small Animals

Hexachlorobutadiene has been shown to be toxic to laboratory mammals.

Acute Toxicity

When given orally, single doses lethal to 50% of the group tested (LD_{50}) ranged from approximately 60 to 600 mg/kg of body weight for rats and from 80 to 160 mg/kg for mice. As well as being related to experimental differences, variations were a function of age and sex. Dermal application and ingestion of HCBD, at levels similar to the ranges noted above, have produced mortality in experiments with rats, mice, guinea pigs, cats and rabbits. A variety of symptoms of acute exposure have been noted in these animals. They include decreased body weight gain, decreased food consumption, increased kidney and liver weight, increased hemoglobin concentration, renal tubule epithelium degeneration, white blood cell breakdown, acidosis, elevated serum nitrogen levels, decreased total blood protein levels, decreased blood and brain sulfhydryl group content, depletion of abdominal fat, decreased red blood cell water permeability, impairment of the immune response, and interference in nerve activity (Schwetz *et al*. 1977; Kociba *et al*. 1977a and b; TSCA/ITC 1978; Harleman and Seinen 1979; Davis *et al*. 1980; USEPA 1980).

The prime target seems to be the kidney. Acutely toxic doses of HCBD damage the straight portion of the proximal renal tubules which are located near the corticomedullary junction. Ammonium transport is affected, plasma urea levels rise, plasma alkaline phosphatase activity increases, and the kidney swells due to an increased water content (Lock and Ishmael 1979, 1981). In addition, liver water content increases, total non-protein sulfhydryl content in the liver decreases, and slight fatty changes are noted histologically (Lock and Ishmael 1981).

Chronic Toxicity

In laboratory studies with rats, chronic symptoms were very clearly exhibited at an oral dose of 20 mg/kg of body weight per day over periods of up to two years. These included decreased weight gain and food consumption; decreased survival time; increased urinary excretion of coproporphyrin; increased kidney weight; renal adenomas and adenocarcinomas, some of which had metastasized to the lung; and increased renal hyperplasia/proliferation (Kociba *et al*. 1977a and b; Schwetz *et al*. 1977).

In a similar type of study over a 13-week exposure period, a no effect level for rats was estimated at 1.0 and 2.5 mg/kg per day for females and males, respectively. At higher levels of exposure (6.3 and 15.6 mg/kg per day) symptoms such as growth inhibition, degeneration of proximal renal tubules, decreased urine concentration ability, increased kidney to body weight ratios, and liver enlargement related to a possible increase in the granular endoplasmic reticulum of the hepatocytes were noted (Harleman and Seinen 1979).

Reproduction

An oral dose of 20 mg/kg of body weight per day over periods of up to two years had no effect on pregnancy and neonatal survival and development. Although the mean weight of weanlings was significantly lower, the absolute difference was small. Over the same period, 2 mg/kg per day produced fewer and less severe symptoms, while at 0.2 mg/kg per day no adverse effects were noted (Kociba *et al*. 1977a and b; Schwetz *et al*. 1977). Doses of 1.0, 2.5, 6.3 and 15.6 mg/kg per day had no effect on reproduction other than lowered weights at birth and weaning of the young of exposed rats (Harleman and Seinen 1979).

A summary of less recent data concerning other species as well as other modes, methods, schedules and durations of application is presented in USEPA (1980). Although most are generally in agreement with the above, there are some data on neurotoxic effects that suggest that the no effect levels could be up to several orders of magnitude lower than the levels of HCBD causing effects

such as those on the kidney (Murzakaev 1967; Poteryaeva 1966; both cited in USEPA 1980).

Mutagenicity, Teratogenicity and Carcinogenicity

Mutagenicity of HCBD was examined in three reversion tests with *Salmonella typhimurium*. Two of the studies reported that it was mutagenic (Tardiff *et al*. 1975 cited in TSCA/ITC 1978; Simmon 1977 cited in Fishbein 1979). The other concluded that although a dose dependent increase in the reversion rate was found, the magnitude was not sufficient to exceed the usual criterion; therefore it was reported as not being mutagenic (Taylor 1978 cited in USEPA 1980).

Teratogenicity has not been examined sufficiently to permit any comment (Poteryaeva 1966 cited in USEPA 1980; Schwetz *et al*. 1977).

Cancerous renal neoplasms attributable to the dietary intake of HCBD were observed in rats after two years of exposure to 20 mg/kg per day. Comprehensive examination did not exhibit any increase in the occurrence of neoplastic lesions in other organs and tissues. Lower dose levels (0.2 to 2 mg/kg per day) did not produce any neoplasms (Kociba *et al*. 1977b). Intraperitoneal injection of HCBD twice weekly for a total of 11 to 13 injections failed to produce a significant increase in the number of lung tumours per mouse, nor was a dose-response relationship observed (Theiss *et al*. 1977 cited in USEPA 1980).

Modes of Action and Interaction with Other Compounds

A decrease in mercapto(SH)-group levels in blood and brain has been noted as a response to chronic ingestion of HCBD (Murzakaev 1967 cited in USEPA 1980; Lock and Ishmael 1979, 1981). As well, compounds containing mercapto, such as mercaptide, cysteine, unithrol and especially 2,3-dimercapto-propane sulfonic acid, are known to be effective antidotes to HCBD poisoning. The actions of HCBD in the mammalian body are probably a function of reactions with compounds of this nature (Mizyukova *et al*. 1973 cited in USEPA 1980). These compounds may facilitate HCBD excretion as well as providing detoxification by conjugation (Davis *et al*. 1980). In general, little is known of the toxic action of HCBD.

Recommended Limit for Livestock Watering

Research on livestock would be necessary before a safe level could be proposed other than by extrapolation from small laboratory mammal data. These data indicated that the organ most severely affected was the kidney, but a number of other effects including a decrease in growth rate were also recorded.

Water for Irrigation

HCBD in Plants and Soil

No information could be found in the literature on the toxicity of HCBD to plants. However, it should be noted that in the USSR it has been applied at a rate of 250 kg/ha in vineyards to control *Phylloxera* on grapes (Vorok'eva 1980). In Germany, it has been applied to soil in which vegetables (potatoes and carrots) were grown, at a rate of 6.17 kg/ha (Fragiadakis *et al*. 1979). Although not specifically studied, the effects on plant growth were not reported in either case.

It has been reported that HCBD stimulates plant growth (Popov 1976). The direct application of HCBD to the seeds of sugar, beet, corn and wheat produced substantial, although species-dependent, stimulation in the growth of both the roots and the aboveground portions. When beans and especially tomatoes and tobacco previously unexposed to HCBD were planted in soil containing HCBD, they exhibited enhanced growth, on the order of 115% to 135% of the controls. The optimum range was from 25 to 100 mg of HCBD per kilogram of soil.

Information on levels of HCBD in plants or plant products is very limited. Hexachlorobutadiene has been detected in wine must and grape juice in the USSR at levels ranging from 0.01 to 0.45 µg/L, but not in fermented wine or pasteurized grape juice (Gorenshtein 1973 cited in IARC 1979). In the United Kingdom, McConnell *et al*. (1975) reported the following HCBD levels: 0.8 µg/kg in tomatoes grown on a reclaimed chemical dump site; 3.7 µg/kg in imported grapes; 0.2 µg/kg in vegetable cooking oil; and 0.2 µg/kg in light ale.

In Germany, vegetable oil margarine and chicken feed (grain) were found to have HCBD levels of 33 and 39 µg/kg, respectively (Kotzias *et al*. 1975). In a study of 20 vegetable samples taken from within 40 km of ten chemical plants where HCBD was a suspected process by-product, Yip (1976) found that none contained detectable levels of HCBD (detection limit of 10 µg/kg). Six of the ten plants were also investigated by Li *et al*. (1976). Yurawecz *et al*. (1976) reported that HCBD was not detectable in spinach samples collected in the lower Mississippi River basin. No Canadian data on HCBD levels in edible plants are available.

The only study in which soil and plant levels of HCBD were simultaneously examined is that by Fragiadakis

et al. (1979). Actual levels of HCBD were not measured; instead, ^{14}C-labelled HCBD was used and HCBD concentrations were estimated from radioactivity measurements. Although it was not reported, it appeared that all of the levels were presented in terms of dry weight. Mean HCBD levels in treated soil were about 60 µg/kg, ranging from about 135 µg/kg in the top 10-cm layer to about 25 µg/kg at 30 to 50 cm below the surface. Levels in the tops and the tubers of potatoes were within this range, but the level in the inedible portion of the roots (approximately 325 µg/kg) was greater than five times the mean soil level and higher than two times the highest soil concentration. Carrots and carrot tops exhibited levels of about 25 to 50 µg/kg, respectively, which were similar to observed soil levels. Based on this scanty evidence it appears that terrestrial plants generally do not accumulate HCBD to levels appreciably above the corresponding soil concentration.

No HCBD levels are known for agricultural fields where HCBD is *not* deliberately applied. In the United States, river bank soil in Louisiana exhibited levels of from 22 to 800 µg/kg, representing 22 to 566 times the concentration in the nearby river (Laseter *et al.* 1976). A wide variety of soil HCBD levels have been found in the vicinity of some plants where chlorinated hydrocarbon solvents are manufactured. At two locations peak levels were 29 and 980 mg/kg within the plant site and 0.15 and 0.34 mg/kg just outside the plant property (Li *et al.* 1976).

Persistence in the terrestrial environment is essentially unknown. Two reports, one Russian and one German, provide some information regarding agricultural applications and the persistence of HCBD in soil. The recorded data are by no means rigorous. Fragiadakis *et al.* (1979) estimated soil HCBD levels on only three occasions (including a calculated value based on the application rate for time zero) over about 15 months. This represents a decay constant of about -0.16 mg/kg per month and for a half-life of about five months, assuming exponential decay. Vorok'eva (1980), over a study period of four years, generated a somewhat larger data base. Interpretation of his data gave a similar indication of decay rate and half-life. These values cannot be construed as accurate, since there were a number of unknown factors, inter- and intra-report variances existed, and assumptions were required for interpretation. They are, however, useful as a rough first approximation of the net effect of physical and biological processes occurring in the soil-plant component of the terrestrial ecosystem.

Recommended Limit for Irrigation Water

There is no significant information on which to base a water quality objective for HCBD in water for irrigation purposes.

Recreation and Aesthetics

Recommended Limit for Recreational Waters

There is no recommended limit for HCBD for recreational waters.

Industrial Water Supplies

Recommended Limit for Industrial Water Supplies

There is no recommended limit for industrial water supplies.

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